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THE SIMULATION OF STOCHASTIC EPIDEMICS IN TWO DIMENSIONS

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1. Introduction

The real purpose of epidemic theory is not to develop interesting and elegant mathematics, though this may be a delightful incidental byproduct, but is to facilitate the practical prevention or control of actual outbreaks of serious contagious disease. This purpose is still a long way from being achieved to any appreciable extent. The developed countries are today free from disasters of the magnitude of the Black Death in the 14th century when perhaps as much as 25 per cent of the population in Europe perished. Nevertheless, widespread epidemics on a massive scale are still common in Africa and the Far East. As the volume and speed of modern travel continue to increase there is an ever growing risk of the transmission of virulent infections to regions where natural immunity may be low though public health control is, for ordinary purposes, more or less adequate. Even within a developed country there are possible dangers from such factors as the appearance of new strains of infectious organisms resistant to standard drugs and antibiotics, or increases in the contact rate between individuals due to greater population densities or changes in social behavior. The current increase in venereal infections in many countries could be a case in point. It follows therefore that it is eminently worth considering in what directions research should proceed in order to have an improved chance of attaining its object.

As with applications to many other fields in biology and medicine, the attempt to develop mathematical theories of epidemics exhibits the usual conflict between insight and realism. Epidemic theory falls into two distinct, though complementary, parts. On the one hand, there is the study of small groups like individual families. From these it is possible, though not often done, to collect detailed data to which relatively realistic models can be fitted, yielding information about such biological or clinical entities as contact rate, length of latent and infectious periods and so forth. However, little can be deduced from this about the spread of infection through a community. The latter requires the special theory of large groups. This provides some insight into the behavior of population outbreaks, with regard to such features as threshold phenomena or the general graphical appearance of "epidemic curves." Unfortunately, even the

237

simplest mathematical models, especially if they are stochastic formulations, present formidable problems of analysis. Any insights obtained therefore are liable to be most true of highly oversimplified models. As soon as an appreciable degree of realism is introduced, especially when geographical spread is involved, the treatment becomes largely intractable. The simplified mathematical theory is worth pursuing because of the hints and leads it may produce, but its limitations should not be forgotten.

It is within this context that more serious consideration should be given to the potentialities of simulating epidemic processes, particularly as the power and availability of automatic electronic computers are constantly increasing.

Most real communities entail the spatial distribution of individuals as an essential ingredient, with susceptibles in the "neighborhood" of an infective being more likely to contract his disease than those that are distant. "Neighborhoods" may, however, be very complex and hard to define, while various forms of complicated social and geographical stratification may also exist. With the aid of modern computer methods of data processing and numerical analysis it seems just within the bounds of possibility that a highly diversified community could be represented by a moderately realistic model and subjected to investigations of a simulation of Monte Carlo type.

We shall look briefly at existing theory, first for deterministic spatial models, and then for the corresponding stochastic versions. Next, we shall examine some new simulation studies of stochastic epidemics in two dimensions, using models which so far are highly oversimplified, but are easily capable of considerable modification and extension. Lastly, we shall discuss the general implications of this type of work to the practical problems facing public health authorities.

2. Deterministic theory

It is always worth examining a deterministic model first, even when we are sure that a stochastic formulation is essential to adequate realism, provided that the inherent limitations are not lost sight of. Stochastic models are invariably harder to handle, and this is specially true of epidemic processes. Approximate treatments may be facilitated if we know in advance what kind of features to look for, and important indications *may* be provided by an analysis of the corresponding deterministic process.

There are various ways in which spatial or topographical elements can be incorporated in an epidemic model. Consider, for example, the following formulation due to D. G. Kendall (see discussion in Bartlett [3]). We assume the existence of an infinite two dimensional continuum of population with a density of σ individuals per unit area. Take any point P surrounded by a small areal element dS. Let the numbers of individuals in this area who are susceptible, infectious or removed be $\sigma x dS$, $\sigma y dS$ and $\sigma z dS$, respectively, where the quantities x, y and z are proportions which sum to unity though they may be functions of time and position. Let the contact rate be β and the removal rate be γ.

Now the basic differential equations defining the process can be written in the form

$$\frac{\partial x}{\partial t} = -\beta \sigma x \tilde{y},$$

(2.1)
$$\frac{\partial y}{\partial t} = \beta \sigma x \tilde{y} - \gamma y,$$

$$\frac{\partial z}{\partial t} = \gamma y,$$

where $\tilde{y}$ is a spatially weighted average of y given by

(2.2)
$$\tilde{y}(P, t) = \iint \lambda(PQ) y(Q, t) \, dS,$$

in which dS is an areal element at Q and $\lambda(PQ)$ is an appropriate nonnegative weighting coefficient. Equations (2.1) are an obvious spatial extension of the usual deterministic general epidemic equations (for example, Bailey [1] equations (4.5)), in which the rate of new infections is made to depend on $\tilde{y}$ rather than y.

A suitable initial condition in the present case is to assume a focus of infection uniformly spread over some small circle centered at the origin. An appreciable amount of mathematical discussion is possible, though the development is not without difficulty. So far as any practical reference is concerned the main result available is the following. Subject to certain conditions, it can be shown that a *pandemic* affecting every part of the plane will ensue if and only if the population density of susceptibles σ exceeds the familiar threshold $\rho = \gamma/\beta$. Moreover, if there is a pandemic, its severity ζ is the unique positive root of

(2.3)
$$\zeta = 1 - \exp(-\sigma \zeta/\rho),$$

meaning that the fraction of individuals eventually contracting the disease will be at least ζ in any area of the plane, no matter how far from the initial focus. We thus have a pandemic threshold theorem corresponding to the well known nonspatial version of Kermack and McKendrick [7].

An alternative model, that is in some ways more specific, has been used by Bartlett [2] to discuss the behavior of recurrent epidemics. Bartlett's treatment is fairly general and involves terms representing the migration of both susceptibles and infectives, as well as the appearance of new infectives. All three of these features can be eliminated for the purpose of the present discussion, and the following simple argument results. Writing x and y for the actual spatial densities of susceptibles and infectives, we assume that the action of infection is local and isotropic. This allows us to write the first two differential equations in the form

(2.4)
$$\frac{\partial x}{\partial t} = -\beta x(y + \alpha \nabla^2 y),$$

$$\frac{\partial y}{\partial t} = \beta x(y + \alpha \nabla^2 y) - y,$$

where $x \equiv x(\xi, \eta, t)$, $y \equiv y(\xi, \eta, t)$ and ξ, η are the spatial coordinates themselves, while $\nabla^2 \equiv \partial^2/\partial\xi^2 + \partial^2/\partial\eta^2$.

In the initial stages of an epidemic x will be approximately constant. The second equation in (2.4) can then be written as

$$(2.5) \qquad \frac{\partial y}{\partial t} = Ay + B\nabla^2 y,$$

where $A = \beta x - \gamma$, $B = \alpha\beta x$. Equation (2.5) is of course a standard diffusion equation with solution

$$(2.6) \qquad y = \frac{C}{2Bt} \exp\left(At - \frac{\xi^2 + \eta^2}{4Bt}\right),$$

the constant C being determined by the initial conditions.

If we consider the total volume of infection outside a circle of radius R, this may be calculated as

$$(2.7) \qquad y_R = \int_{\xi^2+\eta^2 \geq R^2} y \, d\xi \, d\eta$$

$$= 2\pi C \exp\left(At - \frac{R^2}{4Bt}\right).$$

Thus,

$$(2.8) \qquad R = 2(AB)^{1/2}t\left[1 - \frac{\log\left(y_R/2\pi C\right)}{At}\right]^{1/2}.$$

As $t \to \infty$ so $R \to 2(AB)^{1/2}t$. From this it follows that the circle of radius R corresponding to any arbitrary value of y_R grows, for sufficiently large t, at the constant rate $R/t = 2(AB)^{1/2}$. This can be interpreted as the *velocity of propagation* of the disease from the initial focus.

So far as practical consequences are concerned, Kendall's model raises the important issue of pandemic thresholds, and is in fact a first step forward towards a more exact understanding of this type of phenomenon. Bartlett's model, on the other hand, allows one to look more closely at the actual rate of spread of infection. The approximation derived above is, however, only of limited application since we have assumed that the epidemic is still in its early stages with roughly constant x.

More recently, Kendall [6] investigated the deterministic theory of epidemic spread for *linear* communities in one spatial dimension, and discussed the forms of epidemic waves traveling out from a focus. The existence of such waves required the density of susceptibles to be above a certain threshold value. Similar results might be expected for the two dimensional case, but an exact analysis is not yet available.

No doubt the more thorough examination of deterministic models will in due course reveal further important properties. The main point of such work is, however, to see what light may be shed on the behavior of more realistic stochastic versions.

3. Stochastic theory

No general theoretical treatment is yet available for any stochastic analogue of the two dimensional deterministic models described in the previous section, though Morgan and Welsh [8] have recently discussed a rather special kind of stochastic infection process on a lattice. Bartlett [2] has obtained an appropriate partial differential equation for the probability generating functional of a suitable "point" process, but this has so far proved intractable to a general analysis. Some progress was, however, possible with the initial stages of an epidemic when the numbers of susceptibles could be regarded as approximately constant. Bartlett showed that in this special case the behavior of the average number of infectives at any time entailed the propagation of a wave of infection similar to that found in the deterministic situation. No information has been obtained on the general form of the epidemic spread as the stock of susceptibles becomes appreciably depleted.

More recently Neyman and Scott [9] have obtained some very interesting results for a two dimensional model that incorporates a number of realistic features previously neglected. In particular, the number of susceptibles infected by an infective is made to depend on the latter's location, and also an individual infected at any point is allowed to move away and become infectious elsewhere. On the other hand, it is a salient feature of epidemic processes that each new infection diminishes the number of susceptibles at risk. Neyman and Scott's model does not include this aspect, and though approximately valid at the start of an epidemic would become progressively less so as the outbreak built up. How serious the limitation is for the conclusions reached is hard to say. For example, one result, under fairly broad conditions, is that the probability of an epidemic building up in a small community is the same as the probability of an explosive outbreak over the whole of a much larger area. (Compare Kendall's pandemic threshold theorem described in section 2.) This result corresponds with the usual public health view that neglect of sanitary conditions in any part of a country may expose the whole country to danger. One might expect the limitation referred to above to have less effect on this conclusion since in a nonspatial general epidemic we can approximate the early stages by a birth and death process for the population of infectives, ignoring the depletion of susceptibles. And the probability of only a small outbreak is roughly the probability of this process suffering extinction. Another result that needs more careful consideration in relation to certain assumptions made concerns the effect of an immunization campaign on the total size of epidemic. Subject to certain conditions, it appears that the immunization of a random proportion θ of the population would reduce the expected size of epidemic to a value less than $(1 - \theta)/\theta$. This result certainly seems optimistic (as remarked by Neyman and Scott), since it implies that if only ten per cent of the population were immunized the average outbreak would be less than nine, a circumstance that looks improbable if a disease were highly contagious with a small relative removal rate.

As a complementary alternative to theoretical studies, simulation investiga-

tions can be undertaken. Little has been done so far for models incorporating spatial elements. One Monte Carlo study by Bartlett [3] used a 6 × 6 grid of cells. Standard nonspatial continuous time or discrete time processes were assumed within each cell, while a stochastic movement of infectives between cells with a common boundary was adopted. Thus, the spatial element was introduced to a certain extent, though the model was primarily concerned with the behavior of recurrent epidemics in situations where there was a constant accession of new susceptibles.

It was therefore thought worthwhile carrying out a simulation study of a population of individuals all of whom were spread out spatially. This is described in the following section.

4. Simulated epidemics in two dimensions

In this section we describe one of the simplest possible models for a two dimensional epidemic and present a number of results obtained by straightforward Monte Carlo simulations carried out on an electronic computer. As emphasized again later, although this model is very much oversimplified, considerable modifications and extensions could be made with only relatively minor changes in the computer program. Precisely what further work is worth doing is a matter for discussion.

Let us envisage a square lattice of points each occupied by a susceptible individual. It is convenient to regard this community as having finite size. For the present investigation a square boundary was chosen, centered on the origin, given by the lines $x = \pm k$, $y = \pm k$. The total population size n is thus $n = (2k + 1)^2$. Most calculations were performed with $k = 5$, though some were done for $n = 10$. Large populations would need to be explored on a bigger computer than the one actually used, an Elliott 803.

In the present study it was supposed that an epidemic was initiated by the susceptible at the origin becoming infected. This involves a certain amount of symmetry, which is convenient in a first investigation, but not of course essential. We could thus examine the way in which the epidemic spread out from the focus without edge effects due to the boundaries occurring in the initial stages, and ensuring that their influence was symmetrical when they did occur.

A discrete time model of chain binomial type (see Bailey [1] for general discussion and references) was adopted involving two forms, one representing a simple epidemic with no recovery and one a general epidemic with infectives undergoing removal. We first describe the simple epidemic.

4.1. *Simple epidemic with no removal.* Let us suppose, as in ordinary chain binomial theory, that after infection there is a fixed latent period following which the infective becomes highly infectious for a very short period of time. During this infectious phase, ideally contracted to a point, there is probability p of any *susceptible at risk* contracting the disease from the infective in question. But a given susceptible may be exposed to several infectives. If the latter are r

in number, the probability of the susceptible becoming infected is $1 - (1 - p)^r$. In the chain binomial theory of Greenwood or Reed-Frost type an infective is assumed to recover, or at least be removed, immediately after the point of infectiousness. But since in the simple epidemic we have no removal, a convenient assumption is that the infective again becomes highly infectious after a further interval equal to the original latent period, and so on indefinitely. In short, each infective has a series of infectious points, following the initial infection, all separated by intervals equal to the latent period. The epidemic will therefore spread in a series of discrete stages or generations.

Now we also have to decide what individuals are potentially at risk from any given infective. As we have deliberately introduced a spatial element by spreading out the population over a lattice some restriction is evidently needed, otherwise we should simply be returning to a nonspatial model. The most obvious assumption would be to regard only the four nearest neighbors as being at risk (unless already infected). This type of assumption is often used in various kinds of random walk models, and might well turn out to be mathematically tractable. However, there are certain disadvantages. One is that such a restriction might be thought unduly strong, though this is perhaps a small point in a preliminary study. More serious is the limitations involved in using a small computer.

It is easy to see that if the disease is so infectious that $p = 1$, then it will spread in a deterministic manner covering at the gth generation a complete square whose *corners* are at the four points $(\pm g, \pm g)$. As this square has its *diagonals* along the main x and y axes, the edge effects which start occurring when $g = k + 1$ are more elaborate than they would be if the square had its *sides* parallel to the main axes so as to be similarly situated to the population boundaries. Of course we could change the latter by turning the population square through 45°, but this makes the computer program much more complicated if we are fully to utilize a given square array, the elements of which represent the positions of the individuals in the population.

Accordingly, it seemed simpler to assume that the *eight* nearest neighbors to any infective were at risk (unless already infected), as shown in figure 1. With this arrangement, the deterministic spread occurring when $p = 1$ means that at the gth generation a complete square is infected whose *sides* are given by the lines $x = \pm g$, $y = \pm g$. Thus at generation k the population square is completely covered, whereas at generation $k - 1$ none of the susceptibles on the boundary is infected. The edge effects are now very simple in form. The epidemic simply spreads steadily from the focus until the boundary is reached, when it ceases.

Of course we are not specially interested in the case $p = 1$, but for smaller values of p we may expect a probability spread of disease in which some degree of symmetry is retained *vis-à-vis* the boundaries.

The computed simulations were programmed in ALGOL and carried out on the small Elliott 803 computer in the Unit of Biometry, Oxford. The general

scheme of the computations may be briefly outlined as follows. An array $\{a_{ij}\}$, with $i, j = -k, -k + 1, \cdots, 0, \cdots, k - 1, k$, is used to represent the population of individuals. If the individual at the point (i, j) is susceptible $a_{ij} = 0$, if infectious $a_{ij} = 1$. And, initially, $g = 0$ with $a_{00} = 1$, all other a_{ij} being zero. At the gth generation all parts of the array that could have been reached by the epidemic, that is, anywhere in, or on the boundaries of, the square formed by the lines $x = \pm g$, $y = \pm g$, are systematically inspected. If any $a_{ij} = 1$, no change is possible. But if $a_{ij} = 0$, we have a susceptible who may be at risk. All the eight nearest positions must be examined and the sum of the values of
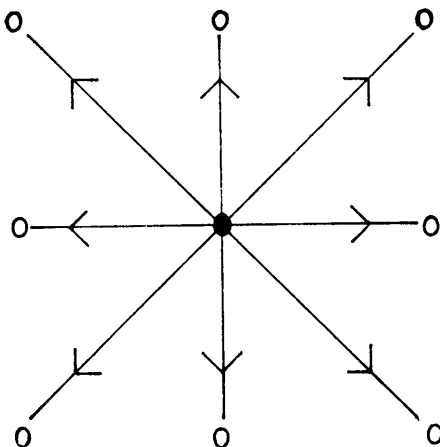

FIGURE 1

Individuals at risk from a given infection.

the relevant a_{ij} formed. Let this be r. If $r = 0$ there are no adjacent infectives. If $r > 0$ then a_{ij} must be changed from 0 to 1 with probability $1 - (1 - p)^r$. An appropriate pseudorandom number is calculated, and the relevant transition performed. The random number algorithm $x_{n+1} = 5x_n(\bmod 2^{35})$ based on Behrenz [4] was used. This is very quick to compute though not entirely satisfactory from a statistical point of view because of appreciable serial correlations between successive terms. It was however considered to be sufficiently accurate for the purpose in hand. We proceed in this way for some arbitrary, but sufficiently large number of generations, in order to complete a single simulation.

In the investigations whose results are described below 25 separate simulations were performed for each epidemic set up with a different value of p. While a larger number of repetitions is desirable, say 100, the results seemed generally useful with many coefficients of variation being of the order of 10 per cent or less. It would be very easy to run much larger numbers of simulations on a big computer using the same ALGOL programs. Also, a more satisfactory random number generator could be inserted in the program.

Now it would be tedious to interpret and wasteful of computer storage

capacity, to try to keep records of the stochastic behavior of the process at each point of the lattice. We may conjecture that some of the symmetry present when $p = 1$ still remains for smaller values of p. In which case it is convenient to amalgamate results for the boundaries of a square of given size. There is bound to be some lack of symmetry at the corners since the risk of infection is always rather less there, but we have chosen to ignore this. When any stage of a particular simulation has been completed, we record the total sum of the *changes* in the a_{ij} values along the boundaries of each square, and also keep a running total of these sums and a running total of the squares of these sums for all simulations to date. At the end of the set of 25 simulations we can calculate means and standard errors for the quantity in question. This quantity is in fact an *epidemic curve*, on the usual definition (see Bailey [1]), for the whole set of individuals along the boundaries of a given square.

Results are shown for the case $k = 5$, that is, an 11×11 square, in figures 2, 3, 4, and 5, with $p = 0.2$, 0.4, 0.6 and 0.8, respectively. The figures shown against the graphs themselves refer to the five available squares. The uppermost graphs (a) in each figure record the average epidemic curve for each square as a whole, while the middle graphs (b) show the *average per individual* in each square. This latter quantity is therefore the epidemic curve relating to the average position in a given square. Finally, the lower graphs (c) give the epidemic curve for the whole population of 121 individuals, and the graphs (d) show the distribution of the completion time, that is, the number of generations it takes for the disease first to infect everyone. The material for a given value of p was produced during a single set of computations, each involving 25 repetitions, averaging about three hours computing time in all.

Although it might have been statistically more satisfactory to exhibit these results in the form of histograms, it would then have been impossible to superimpose the several curves without confusion. The frequency polygon method was therefore preferred.

The (a) curves in each figure show how the epidemic spreads more rapidly as p increases, and how the magnitude of the epidemic's effect is greater at greater distances from the focus, though of course more time is required for a build up the further out we go. When p is small the curves are relatively flatter and the build up takes longer than when p is large.

Of more immediate interest are perhaps the (b) graphs which, as already mentioned, show in effect the epidemic curves relating to an average individual on a given square. The curves can also be regarded as frequency distributions since the areas under them must all be unity. These distributions are therefore more directly comparable with one another and constitute one way of representing the advancing epidemic wave. It will be seen that the wave appears, in general terms, to progress at a more or less steady rate, though its effect is more spread as we move further from the focus, at least for small p. When p is large, for example, 0.8, the waves are almost identical in form irrespective of the distance from the origin. And, of course, when $p = 1$ the spread is entirely

deterministic with exactly one new case appearing per point at precisely the gth generation for all points on the square $x = \pm g$, $y = \pm g$.

The more or less linear spread of disease can also be seen in the lower (c)
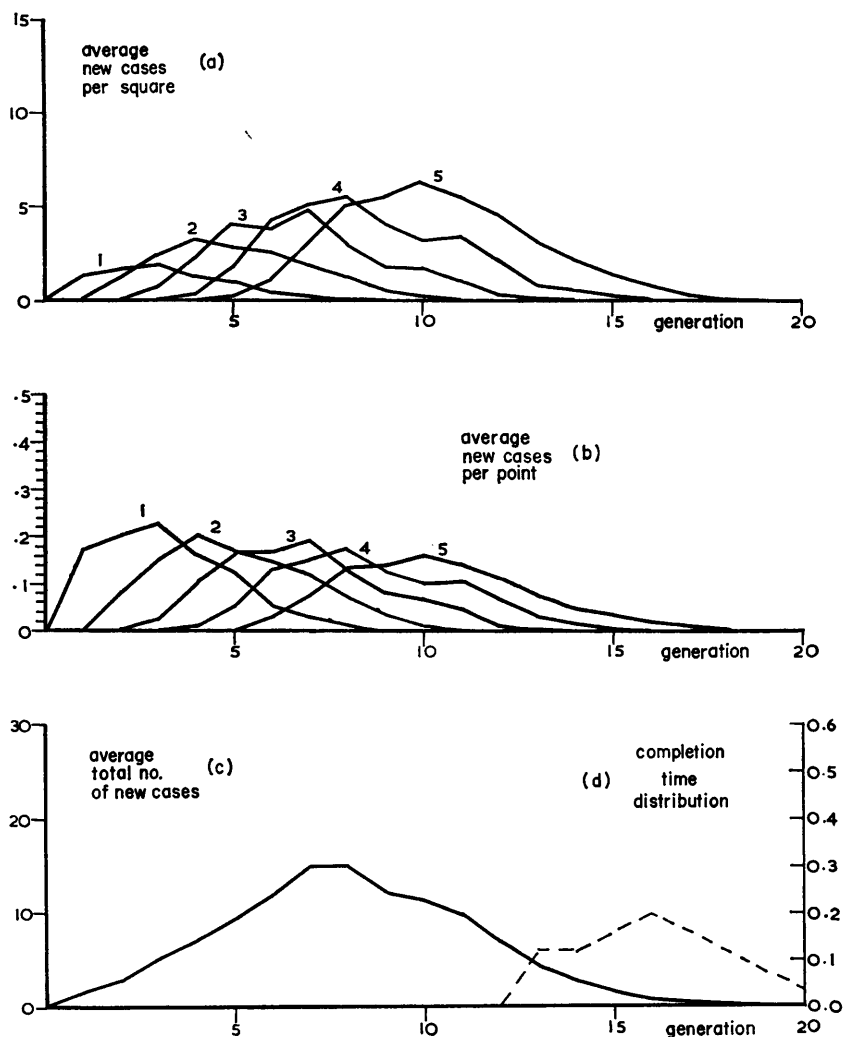

FIGURE 2

Simple epidemic on an 11 $\times$ 11 square with $p = 0.2$.

graphs which show epidemic curves for the whole population. At least, the average growth is linear until about the sixth generation when edge effects begin to appear. As p increases the epidemic curve drops more sharply. When $p = 0.2$ the fall is slower than the rise, but with $p = 0.8$ the cutoff becomes

quite steep, due to the epidemic's reaching the outer boundary with a higher degree of probability. Again, with $p = 1$ the cutoff would be completely vertical. The reason for the linear rise is presumably that in the rather restricted model
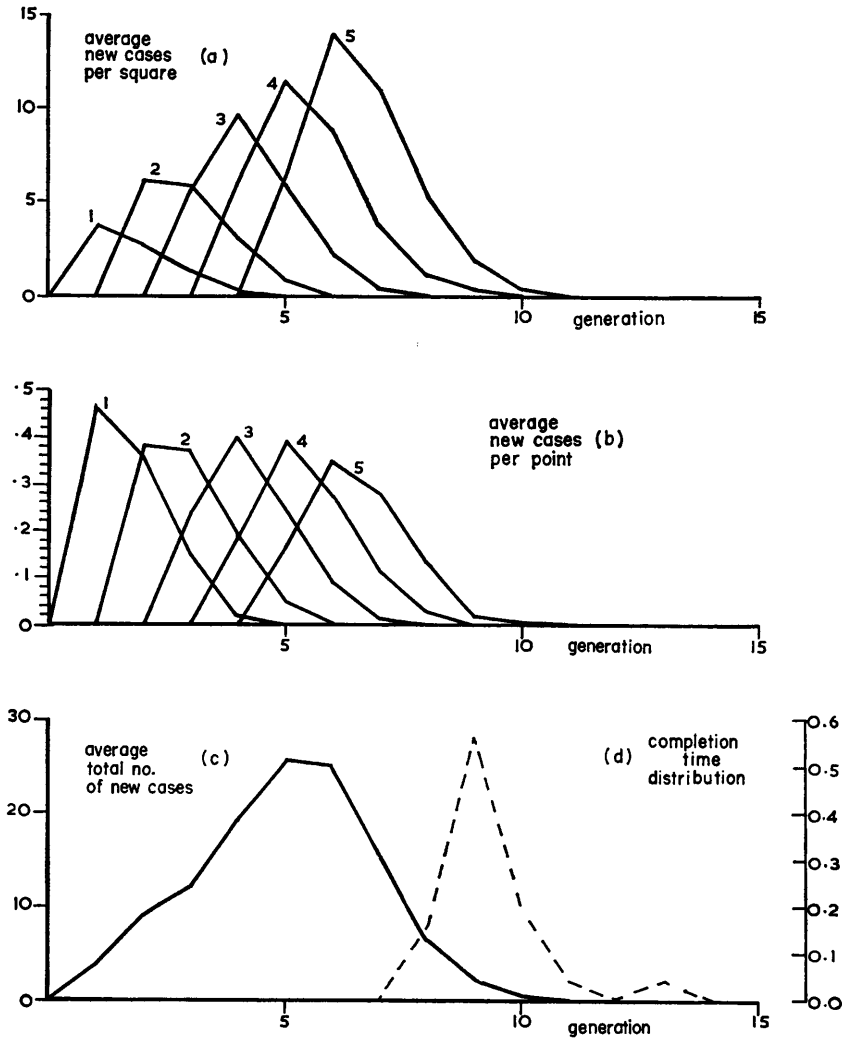

FIGURE 3

Simple epidemic on an 11×11 square with $p = 0.4$.

adopted each infective is in contact with only a small number (eight) of other individuals, and there is not the same opportunity for the probability of infection to rise to the values obtained when there is full homogeneous mixing.

It is not clear whether anything useful can be deduced from the completion
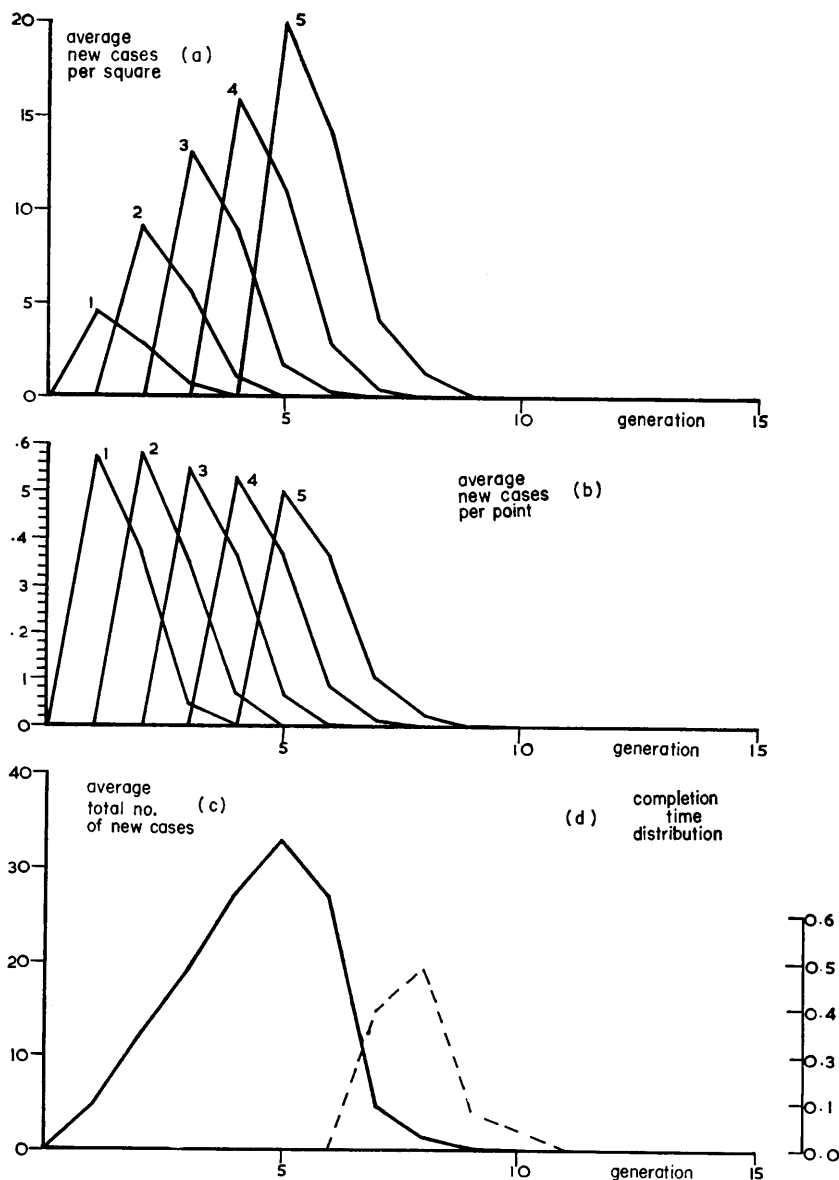
FIGURE 4

Simple epidemic on an 11 × 11 square with $p = 0.6$.

time graphs at (d). Since the whole of each curve is based on only 25 observations, sampling fluctuation is large. But it can be seen that completion time becomes shorter and less variable as the disease becomes more infectious.

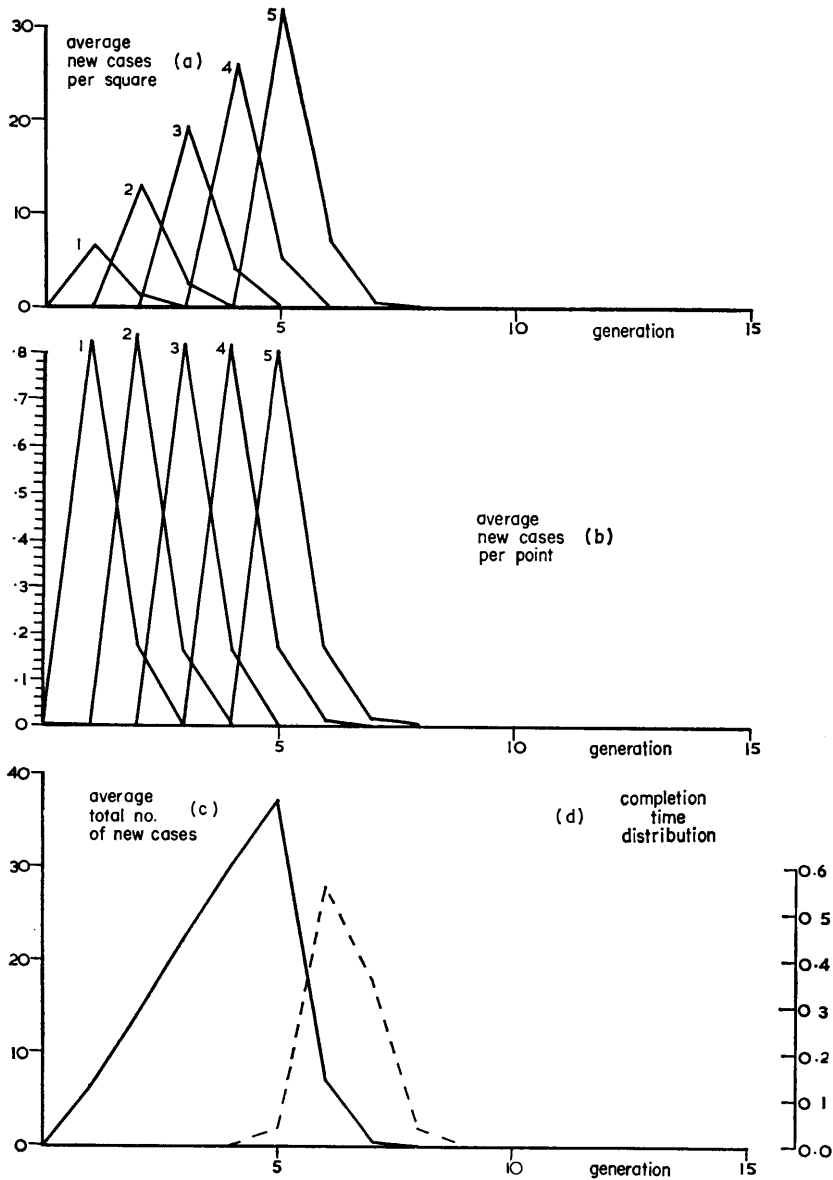A number of simulations were also run for a 21 × 21 square with $k = 10$.

FIGURE 5

Simple epidemic on an 11 × 11 square with $p = 0.8$.

The general conclusions are rather similar to those above, though we can of course follow the progress of the epidemic for a greater number of generations before edge effects predominate. As a single example, let us consider the epidemic curve for $p = 0.6$ shown numerically in table I. It is evident that for the

TABLE I

Epidemic Curve for a Simple Epidemic in a
Whole Population Spread Out Over a
21 × 21 Lattice with $p = 0.6$

Generation	Average No. of New Cases
1	4.4
2	11.8
3	18.6
4	26.7
5	33.1
6	41.2
7	48.5
8	56.0
9	63.5
10	72.2
11	40.1
12	18.1
13	5.1
14	0.6
15	0.2

first ten generations there is an almost constant increase of about 7.5 new cases per generation, after which the epidemic curve falls away rather sharply as before.

4.2. *General epidemic with removal.* The simple epidemic model of the previous section can readily be extended to cover the more general case when removal of some kind is envisaged. We merely have to adopt the familiar chain binomial assumption that after the first occurrence of infectiousness, the infective in question ceases to be infectious and enters a third state, represented in the simulations by $a_{ij} = 2$, say. Alterations required to the previous computer program are minimal. The set of results corresponding to the simple epidemics described above are shown in figures 6 to 9 for the same range of values of p, though the distributions of completion time are now omitted and the distributions of total epidemic size are considered in the separate table II. The total computing time for each value of p was about six hours.

For $p = 0.4$, 0.6, and 0.8 it has been possible to use the same scales as for the simple epidemic, but for $p = 0.2$ large changes were necessary. This corresponds to some kind of a threshold between $p = 0.2$ and $p = 0.4$.

Comparing figure 6(a) with figure 2(a) for instance, we see how in the general case the curves present quite a different appearance. The average number of new cases per square now falls off with distance instead of conversely, and all numbers are absolutely much less than in the simple epidemic. It is clear that we are now dealing with a small local outbreak only, whose presence is felt less and less as we go farther from the focus. The point is made even more obvious by comparing the "point" epidemic curves of figure 6(b) with figure 2(b). In the general
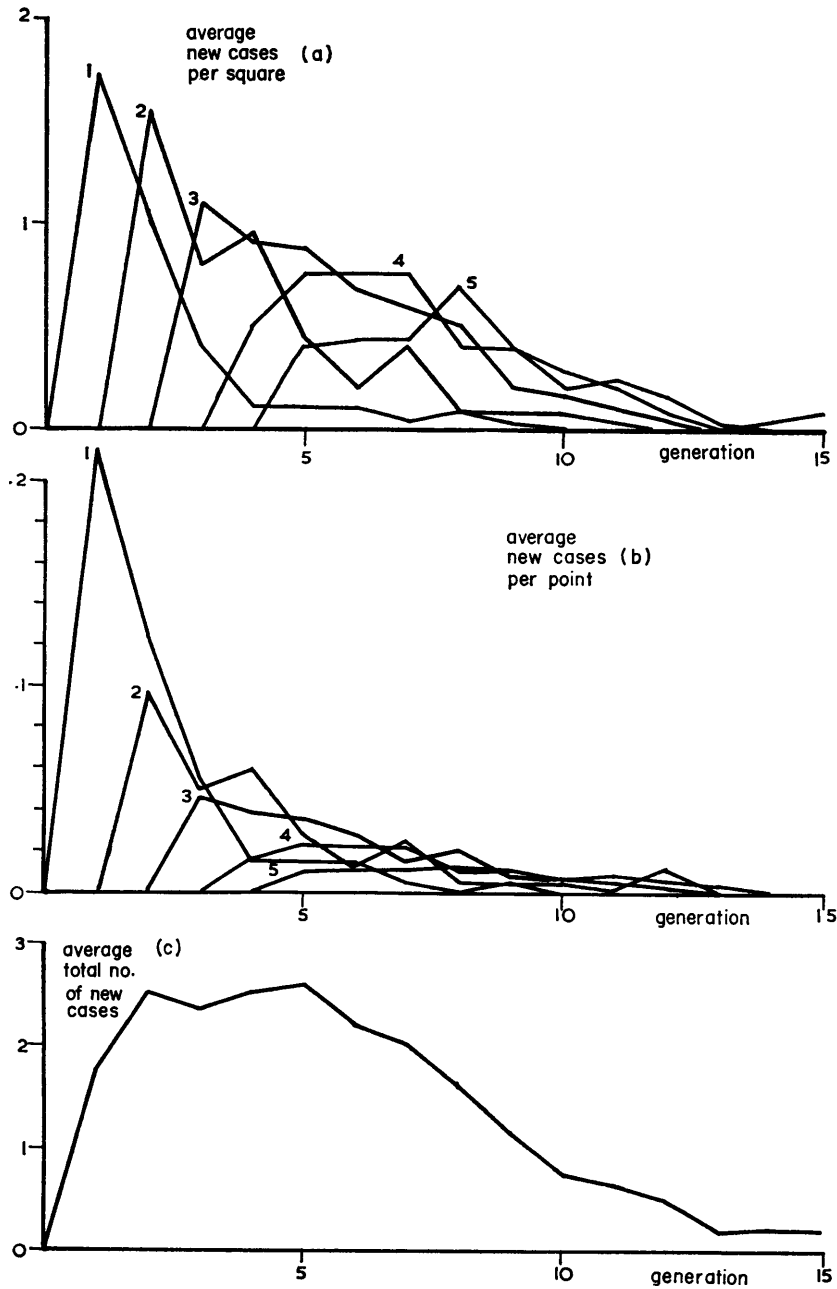
FIGURE 6

General epidemic on an 11 × 11 square with $p = 0.2$.

epidemic we have waves traveling from the center that are rapidly damped out. Similarly, figure 6(c) shows the existence of quite a small outbreak com-
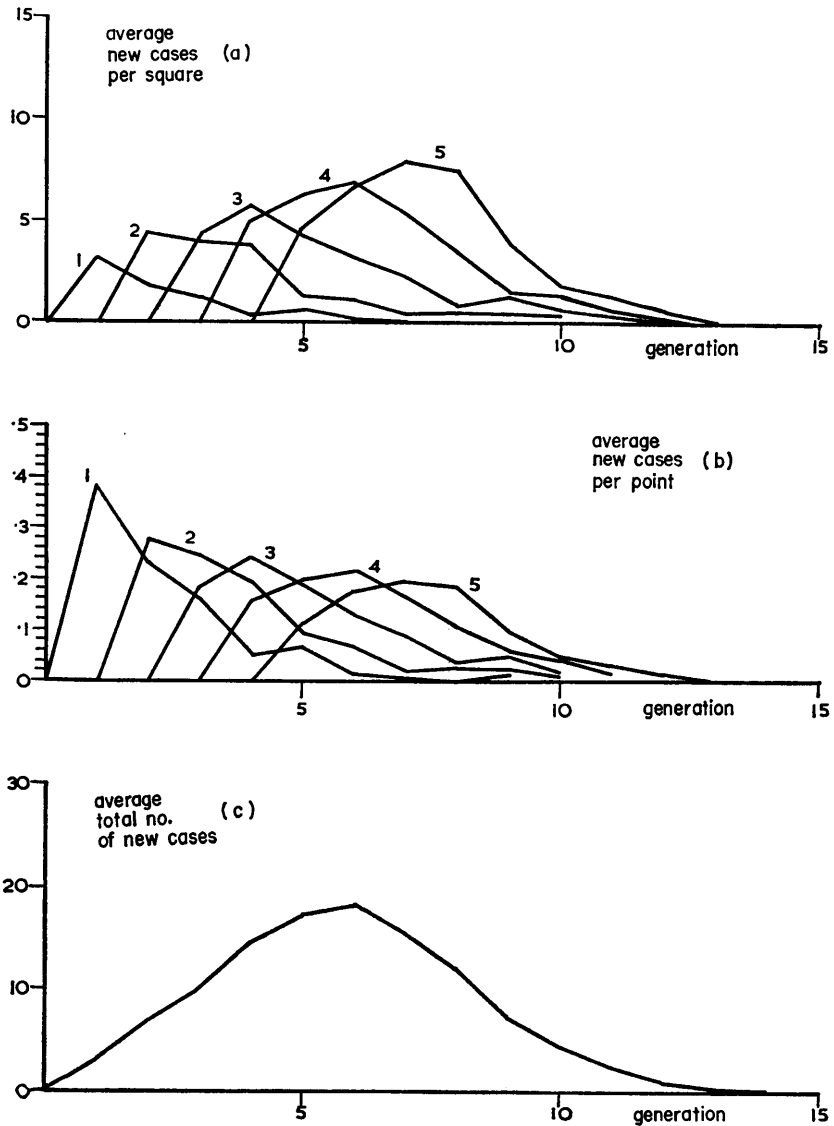

FIGURE 7

General epidemic on an 11 × 11 square with $p = 0.4$.

pared with figure 2(c) when we take into account a tenfold difference in the vertical scales.

When we come to look at figure 7 and figure 3, we find that for $p = 0.4$ the
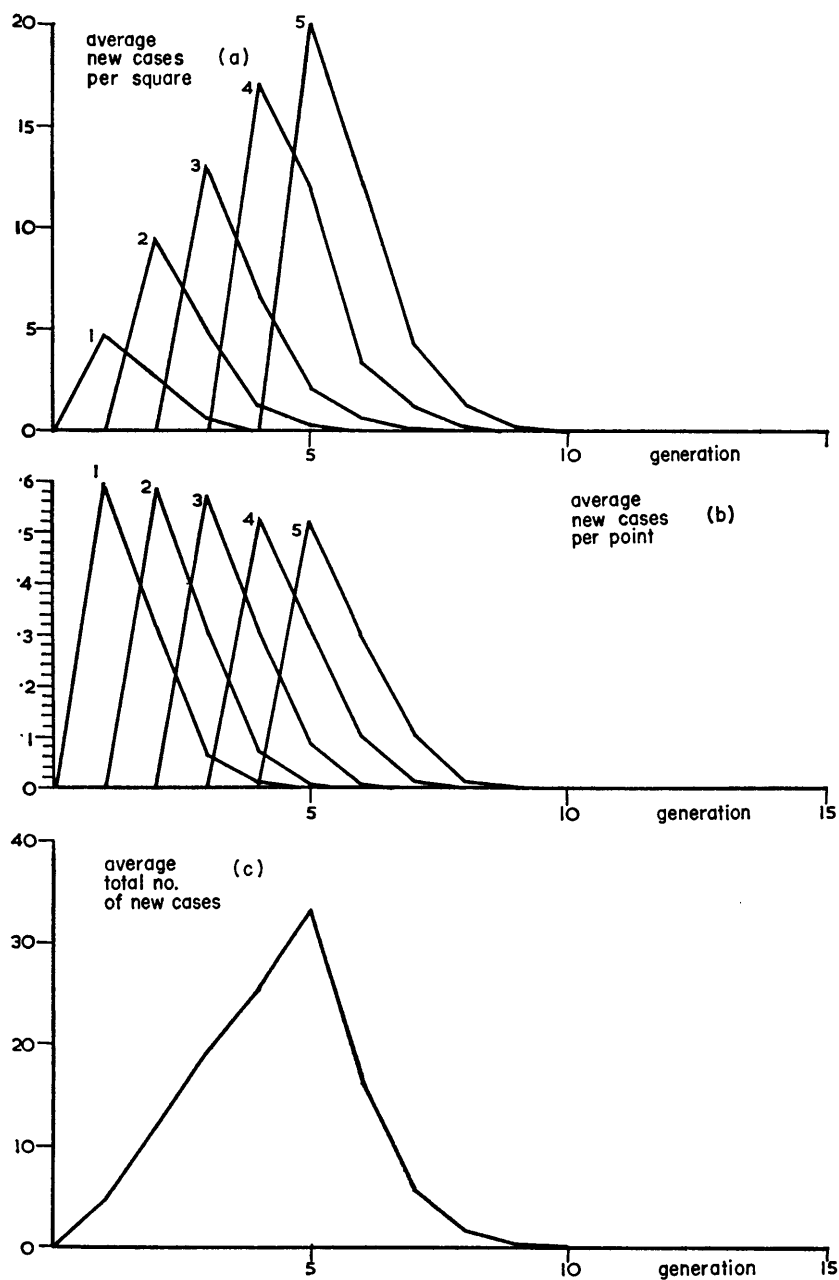
FIGURE 8
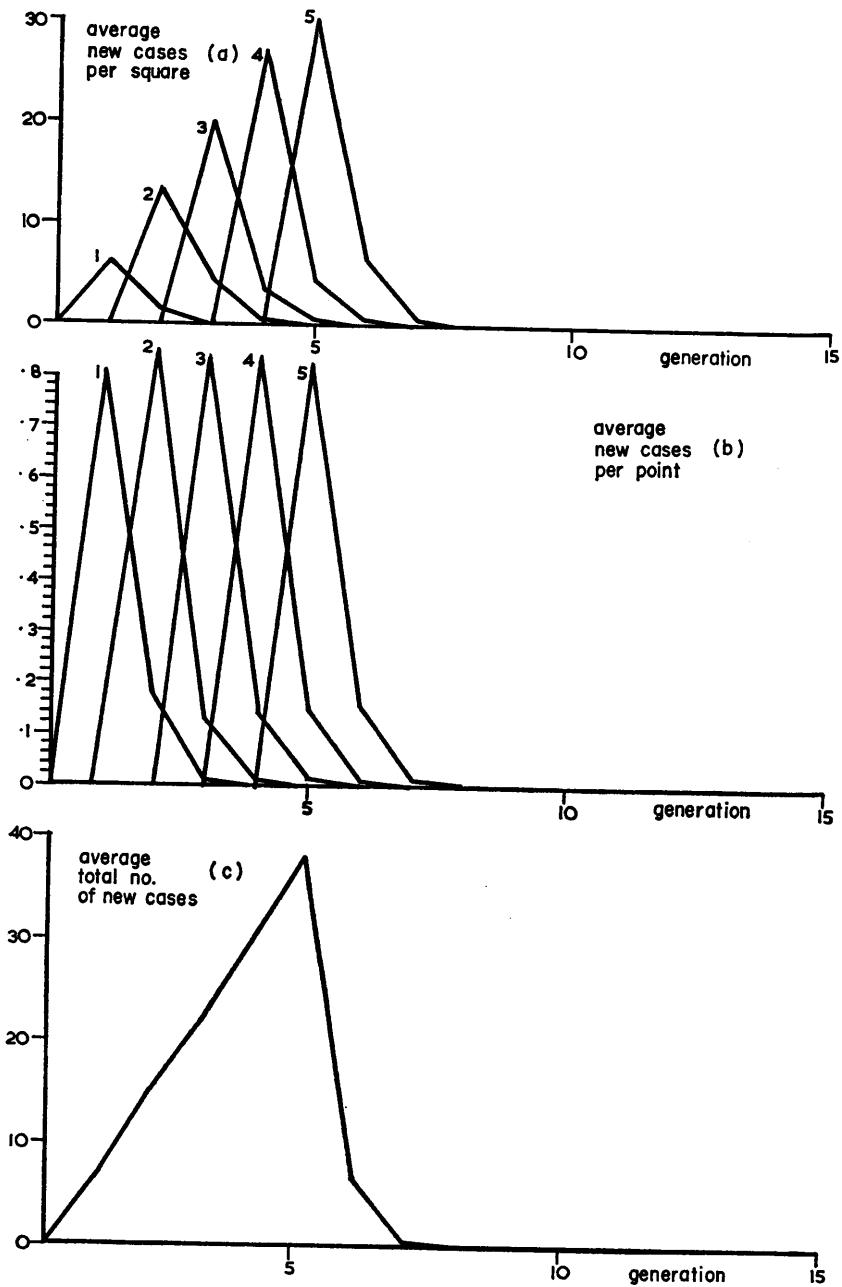
General epidemic on an 11 × 11 square with $p = 0.6$.

FIGURE 9

General epidemic on an 11×11 square with $p = 0.8$.

differences are much less marked. The general epidemic is now definitely spreading in an explosive manner, though somewhat more slowly and diffusely than in the simple epidemic with no recovery. For larger values of p the differences between the general and simple types of epidemic become progressively less marked so far as epidemic curves and epidemic waves of advance are concerned.

The range between $p = 0.2$ and $p = 0.4$ obviously deserves closer attention, and a series of simulations were carried out at intervals of 0.04 with special reference to the distribution of the total size of epidemic. These distributions, fairly coarsely grouped, are shown in table II. Although the samples are all

TABLE II

DISTRIBUTIONS OF TOTAL EPIDEMIC SIZE FOR DIFFERENT VALUES OF p

Total No. of New Cases	Values of p				
	.20	.24	.28	.32	.36
0– 20	13	10	8	2	0
21– 40	6	4	1	1	0
41– 60	5	6	3	2	0
61– 80	1	2	8	4	1
81–100	0	3	5	8	3
101–120	0	0	0	8	21
Total	25	25	25	25	25

rather small, the steady shift in distribution with changes in p is immediately apparent. When $p = 0.20$, just over half the epidemic sizes were 20 or less in a total population of 121. At $p = 0.24$ there are strong indications of a spread over the whole range, while by $p = 0.32$ the concentration is building up at the top end. At $p = 0.36$, over 80 per cent of epidemics have a total size of more than 100. It would be interesting to examine the precise form of this change over from one extreme distribution to another, using a much larger number of simulations for greater accuracy.

5. The role of simulation

Considerable emphasis was placed in the introduction on the importance of theoretical epidemic studies having some practical relevance. We saw in section 2 how some preliminary insights could be obtained into the behavior of epidemics incorporating spatial elements by means of deterministic models. But when we examined more realistic stochastic models in section 3 it seemed that these formulations were either intractable, or could be managed only when confined to the opening stages of an epidemic, or could be developed only if fairly severe simplifications were assumed. Section 4 on the other hand dealt with simulation of some very simple spatial models, in which the limitations were of a different kind. It is highly pertinent, therefore, to ask in what way

such simulation studies can be regarded as having practical applications, and as providing useful results that cannot be obtained by purely mathematical manipulations of the relevant models.

As mentioned at the beginning of section 4, although the models subsequently discussed were considerably oversimplified they were readily capable of extensive development and modification by means of comparatively small changes in the computer programs used. Thus we assumed that individuals were permanently attached to the points of a square lattice, though able to infect any of their eight nearest neighbors. This might be interpreted as permitting some degree of movement, at least sufficient to result in a degree of contact with nearest neighbors. There is no reason why we should not introduce the possibility of more distant individuals being infected, the chance of infection diminishing perhaps with distance according to some assumed law. This means that, when at any stage we are inspecting the array $\{a_{ij}\}$ and find that a particular $a_{ij} = 0$, that is, the point (i, j) is occupied by a susceptible, we should have to search a larger area around the point (i, j) for possible infectives than envisaged in the simple study described. More computer time would be required but no new principle is involved. Indeed, if required, all kinds of heterogeneous spatial structures could be built into the model. Moreover, additional stages might be introduced into the fundamental stochastic process so as to relax the assumptions of constant latent period and point infectiousness.

How far can one go in developing extensions before even the largest existing computer is inadequate is difficult to say. Perhaps a more important question at the present time is what would be practically useful if the computations could be performed in reasonable periods of time. There are two obvious possibilities.

First, far more highly structured models could be developed that contained representations of a very large number of realistic features. The probable consequences of a variety of public health measures such as immunization campaigns, the use of quarantining and restriction of public movements, and so forth, could then be ascertained by reference to models that, though hypothetical, were realistic and typical in the sense of incorporating a large number of actual clinical, biological, social or geographical features. The general insights obtained from such work might be very much more powerful than the elementary notions of thresholds at present available. They might also usefully be combined with the application of operational gaming methods to public health training programs. These have been employed, for instance, by the California Department of Public Health together with the Systems Development Corporation [5], and entail the construction of a simulated city called "Epiville."

Secondly, it might be possible to construct models to represent highly diversified actual communities. In this way the epidemiological status of these communities could be tested in advance of any real outbreak by performing appropriate simulations. Alternatively, the models might be used to assess actual outbreaks of serious disease by facilitating the estimation of biologically im-

portant parameters such as contact rates, removal rates, and so forth. From here one might hope to be able to construct the future development of the outbreak in probability terms, indicating a range of possible consequences if various alternative steps were taken leading to a reduction of contact rates or an increase of removal rates, for example. The results of such calculations might well indicate what kind of public health intervention would be most effective.

These ideas are of course highly speculative, but indicate some of the directions in which further research might proceed.

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